Isofol Medical AB

Corporate Presentation

March 2026

ISOFOL

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iSOFOL

Isofol in short

- Oncology-focused biotech company listed on Nasdaq Stockholm
- Our goal is to make today's and tomorrow's cancer treatment better with **arfolitixorin**, a next-generation folate drug candidate designed to replace leucovorin and enhance the efficacy of standard 5-FU-based treatments for solid tumors
- Currently conducting a phase Ib/II trial in metastatic colorectal cancer, the 3rd most common form of cancer

ISOFOL

Isofol’s value creation rests on three solid pillars

01

High unmet medical need

02

High-potential drug candidate

03

Large market opportunity

ISOFOL

Isofol’s value creation rests on three solid pillars

01

High unmet medical need

Arfolitixorin aims to potentiate 5-FU-based chemotherapy – todays and tomorrow’s standard treatment for several types of cancer where better treatments are urgently needed

02

High-potential drug candidate

03

Large market opportunity

ISOFOL

Arfolitixorin aims to potentiate 5-FU-based chemotherapy, a standard treatment for several types of cancer with high unmet medical need

5-FU

The chemo basis of cytostatic combinations (for example FOLFOX)

+

Folate

Added to enhance the effect by delivering MTHF to tumors (today: leucovorin)

arfolitixorin
next-gen folate

Used in various regimens for treating for example
colorectal, gastric, pancreatic, esophageal and gastroesophageal, head and neck cancers.
Response rates (ORR) of the currently used F-FU-based treatment combinations are typically below 50%.

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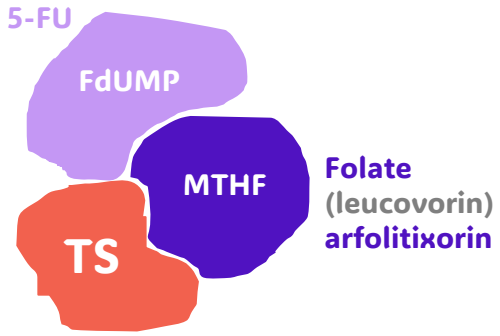
Mode of Action (simplified): 5-FU + folate inhibits the TS enzyme to kill tumor cells



- Target: Intratumoral inhibition of TS (thymidylate synthase) – an enzyme essential for tumor cell growth
- The standard chemotherapy 5-FU is swiftly after administration converted to FdUMP
- FdUMP binds to TS to inactivate it, forming an unstable binary complex where TS can break free and become active again
- High concentrations of MTHF (active metabolite of folate) stabilize TS+FdUMP+MTHF in a ternary complex

- The stable ternary complex inhibits TS effectively, resulting in interrupted tumor cell DNA synthesis and repair

- Anti-tumor effect

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Arfolitixorin has potential across several cancer types treated with 5-FU-based chemotherapy, starting with metastatic colorectal cancer (mCRC) as the lead indication

Colorectal cancer is a major medical problem worldwide because of its high incidence and low survival rates among patients with advanced/metastatic disease:

1 900 000

people are affected annually

3rd most common cancer

900 000

patient deaths per year

2nd most common cause of cancer death

86 %

of patients with metastatic disease (mCRC) die within five years

→ **<50% of patients respond to today's standard treatment, and overall survival is low.**

→ **Improving outcomes of first line therapies would have a high impact on a large patient group.**

Ref: Intl Agency for Research on Cancer, World Health Organization, Accessed 30 April 2025. https://gco.iarc.who.int
American Society of Clinical Oncology (ASCO) Cancer.Net. Accessed 12 March 2024. https://www.cancer.net/cancer-types/colorectal-cancer/statistics.

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5-FU combined with folate is the backbone treatment for 95% of all mCRC patients, today as well as tomorrow.

MSS/pMMR (95% of patients)		MSI-H/dMMR (5%)
RAS/BRAF mutant	**RAS/BRAF wild-type**	
Patients elibible for intensive tx (95%) FOLFOX/FOLFIRI/ CAPEOX +/- bevacizumab or FOLFOXIRI +/- bevacizumab	Right-sided: FOLFOX/FOLFIRI/CAPEOX/FOLFOXIRI +/- bevacizumab	Keytruda Opdivo ± Yervoy
Patients inelibible for intensive tx (5%) Capecitabine + bevacizumab or 5-FU + folate + bevacizumab	Left-sided: FOLFOX/FOLFIRI +/- cetuximab +/- panitumumab	

Addressable patient groups for arfolitixorin
(and the patient groups included in the ongoing trial)

- **Low competition:** Many drugs are in development for mCRC, all intended to be used either as an **add-on** to 5-FU+folate or in **later lines of therapy.** Arfolitixorin is currently the only new drug in development for first-line use designed to replace leucovorin.
- **Lasting market opportunity:** 5-FU combined with folate will remain the backbone treatment for mCRC for the foreseeable future.

Consensus among clinical experts that 5-FU+folate will remain the mainstay of treatment:

"It is inconceivable to think that 5-FU backbone will not be the mainstay care, it is a very effective drug within our treatment landscape"

"5-FU is the most effective drug in this disease, I don't think it will ever go away ever"

"I think we are going to get smarter and find more actionable mutations, but I do not think this will impact the 1L SoC"

"There isn't anything that comes close to chemo or has a response rate high enough to replace the chemo backbone. 5-FU will remain the pyrimidine of choice in multi-agent chemo regimens in 1L"

Ref: Market research conducted by Back Bay Life Science Advisors in November 2024.

Folates are used in 9 out of 10 mCRC regimens – creating a large opportunity for value creation

- Folates are used in most 5-FU-based treatment regimens for mCRC, such as FOLFOX, FOLFIRI and FOLFOXIRI
- Folates (folinic acid) are sold under different names, the most common one being *leucovorin*
- **Arfolitixorin could potentially replace the currently used folic acid across all these regimens**

Regimen*	Irinotecan	Oxaliplatin	Leucovorin¶	Fluorouracil/capecitabine	Schedule
FOLFIRI[1]	180 mg/m^2 day 1		400 mg/m^2 over two hours day 1	Fluorouracil 400 mg/m^2 bolus day 1, followed by 2400 to 3000 mg/m$^{2\Delta}$ over 46 hours, continuous infusion	Every two weeks
Douillard regimen[2]	180 mg/m^2 day 1		200 mg/m^2 leucovorin over two hours days 1 and 2 before fluorouracil	Fluorouracil 400 mg/m^2 bolus then 600 mg/m^2 over 22 hours days 1 and 2	Every two weeks
FOLFOX 4[3]		85 mg/m^2 day 1	400 mg/m^2 over two hours days 1 and 2 before fluorouracil$^{\lozenge}$	Fluorouracil 400 mg/m^2 bolus, then 600 mg/m^2 over 22 hours days 1 and 2	Every two weeks
FOLFOX 6[1]		100 mg/m^2 day 1	400 mg/m^2 over two hours day 1	Fluorouracil 400 mg/m^2 bolus day 1, followed by 2400 to 3000 mg/m$^{2\Delta}$ over 46 hours, continuous infusion	Every two weeks
Modified FOLFOX 6[4,5]		85 mg/m^2 day 1	350 mg total dose over two hours day 1	Fluorouracil 400 mg/m^2 bolus day 1, followed by 2400 mg/m^2 over 46 hours	Every two weeks
FOLFOX 7[6]		130 mg/m^2 day 1	400 mg/m^2 over two hours day 1	Fluorouracil 400 mg/m^2 bolus, then 2400 mg/m^2 over 46 hours	Every two weeks
Modified FOLFOX 7[7] (Optimox)		100 mg/m^2 day 1	400 mg/m^2 over two hours day 1	Fluorouracil 3000 mg/m^2 over 46 hours	Every two weeks
Modified FOLFOX 7[8] (CONcePT)§		85 mg/m^2 day 1	200 mg/m^2 over two hours day 1	Fluorouracil 2400 mg/m^2 over 46 hours	Every two weeks
XELOX[5]		130 mg/m^2 day 1		Capecitabine 1000 mg/m^2 orally twice per day on days 1 to 14	Every three weeks
FOLFOXIRI[9]	165 mg/m^2 day 1	85 mg/m^2 day 1	400 mg/m^2 leucovorin over two hours day 1	Fluorouracil 3200 mg/m^2 over 48 hours	Every two weeks

* All doses shown are for intravenous (IV) administration, except capecitabine.
¶ Leucovorin doses given for the d,l racemic mixture.
Δ 2400 mg/m^2 dose is commonly used.
◊ The original trial report indicated a leucovorin dose of 200 mg/m^2 daily, but this was an error, and the correct dose used in the protocol was 400 mg/m^2 (R Goldberg, personal communication).
§ FOLFOX 7 was administered with bevacizumab (5 mg/kg every two weeks) in the CONcePT trial.

Ref: Clark, J; Sanoff, H. Initial systemic therapy for metastatic colorectal cancer. In: UpToDate, Atkins M (Ed), Wolters Kluwer. (Accessed on Oct 18, 2025.)

iSOFOL

Isofol's value creation rests on three solid pillars

01

High unmet medical need

02

High-potential drug candidate

Arfolitixorin is the first and only direct-acting folate, designed to enhance 5-FU efficacy and improve outcomes of standard treatments across multiple cancer types. It has shown promising results in earlier studies.

03

Large market opportunity

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Arfolitixorin's key advantage: Bypassing the metabolic activation steps required by today's folinic acid drugs

Arfolitixorin
(active drug)

Direct-acting: the active metabolite [6R]-MTHF in itself

resulting in several-fold higher levels of MTHF in tumor tissue[1], and an efficacy that could increase with the dose[2]

Effect

Aim: Improved clinical outcomes

Standard-of-care
Leucovorin
(prodrug)

Multi-step conversion to MTHF driven by endogenous enzymes

Effect

1) Wettergren Y et.al. A pharmacokinetic and pharmacodynamic investigation of Modufolin® (arfolitixorin) vs Isovorin® after single-dose IV administration to patients with colon cancer: a randomized study. Cancer Chemother Pharmacol. 2015;75(1):37–47. doi:10.1007/s00280-014-2611-9n. 2) According to preclinical studies, cf. next slides

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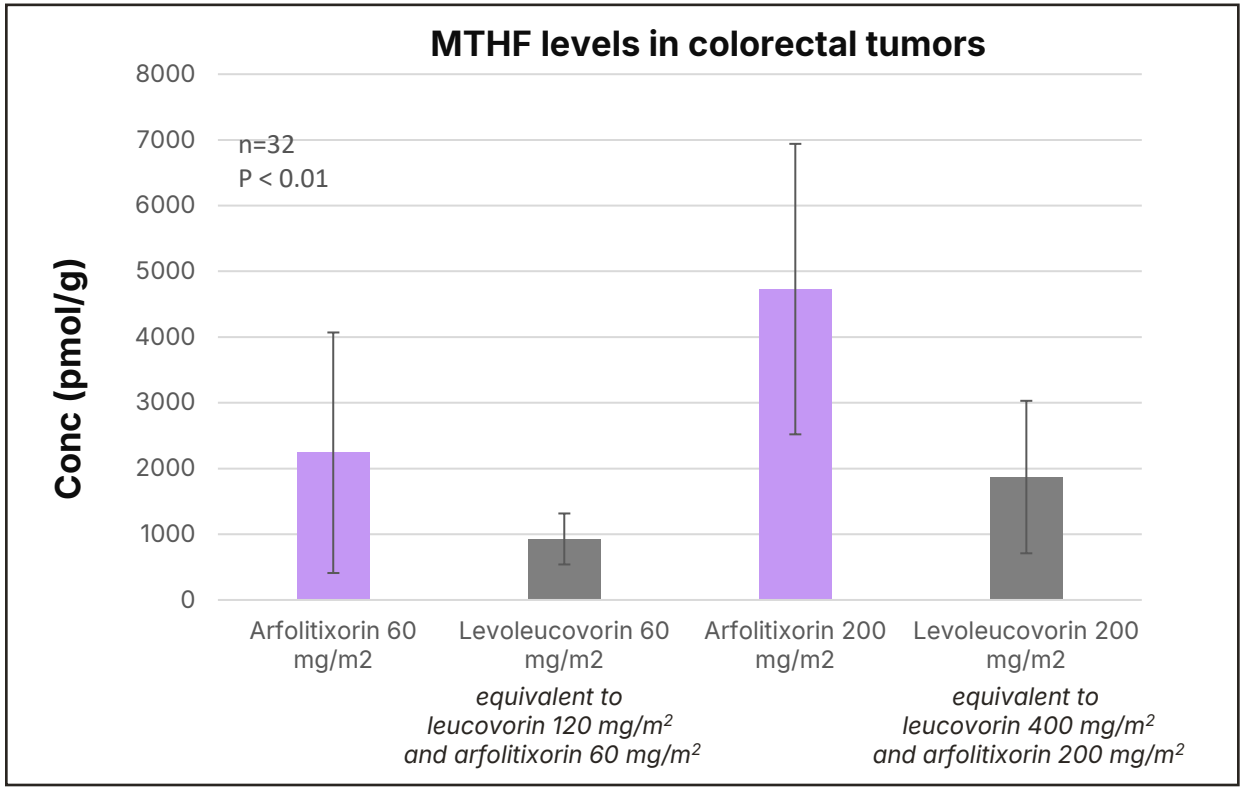
Higher concentrations: Arfolitixorin gives several-fold higher levels of MTHF in tumors, optimizing conditions for TS inhibition



- PK/PD data from the randomized Phase I/II study ISO-CC-002 demonstrate significantly increased MTHF levels in mCRC tumors from patients treated with arfolitixorin as compared to equimolar doses of levoleucovorin*
- Significantly higher levels of MTHF were also observed in the arfolitixorin 200 mg/m^2 dose cohort in comparison to the arfolitixorin 60 mg/m^2 dose cohort. This suggests that elevated doses of arfolitixorin could create conditions for an increased tumor-killing effect by further elevating the intracellular MTHF concentration.

Ref: Wettergren Y, et. al. A pharmacokinetic and pharmacodynamic investigation of Modufolin® (arfolitixorin) vs Isovorin® after single-dose IV administration to patients with colon cancer: a randomized study. Cancer Chemother Pharmacol. 2015;75(1):37–47. doi:10.1007/s00280-014-2611-9

*) Levo-leucovorin contains only the L-isomer, so it delivers the same pharmacologic activity at half the dose as leucovorin that also has the D-isomer

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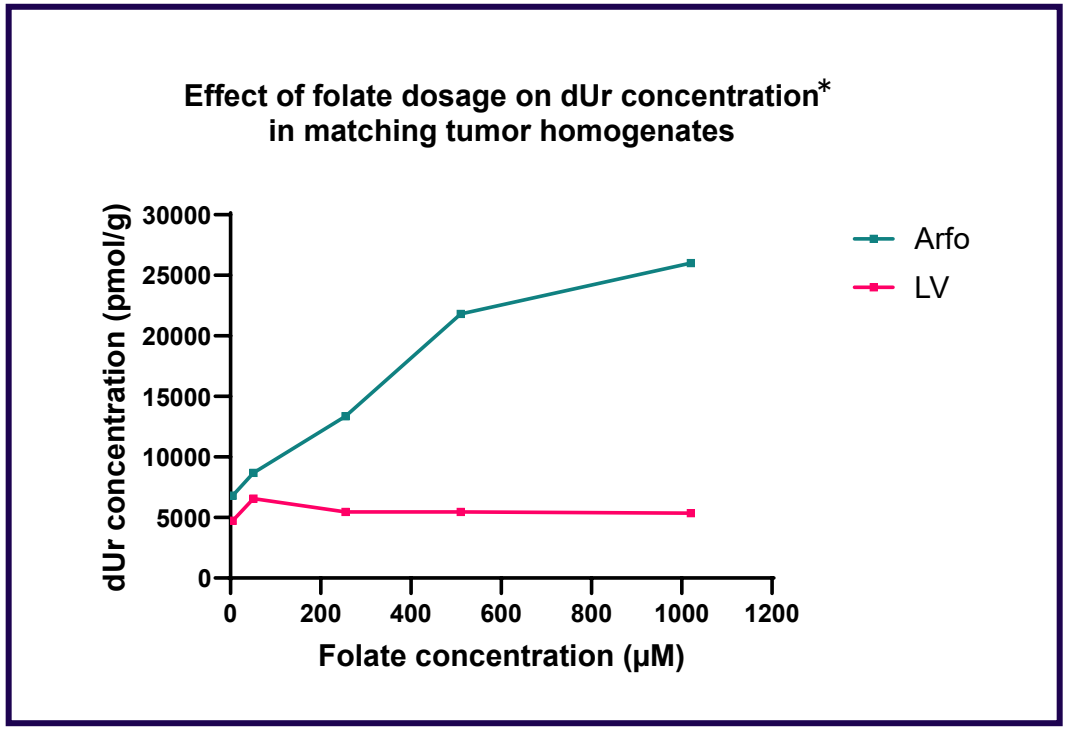
Dose-response: Studies indicate that arfolitixorin's efficacy increases with dose, setting it apart from leucovorin (1/3)



*) dUr concentration is a surrogate marker for TS inhibition.

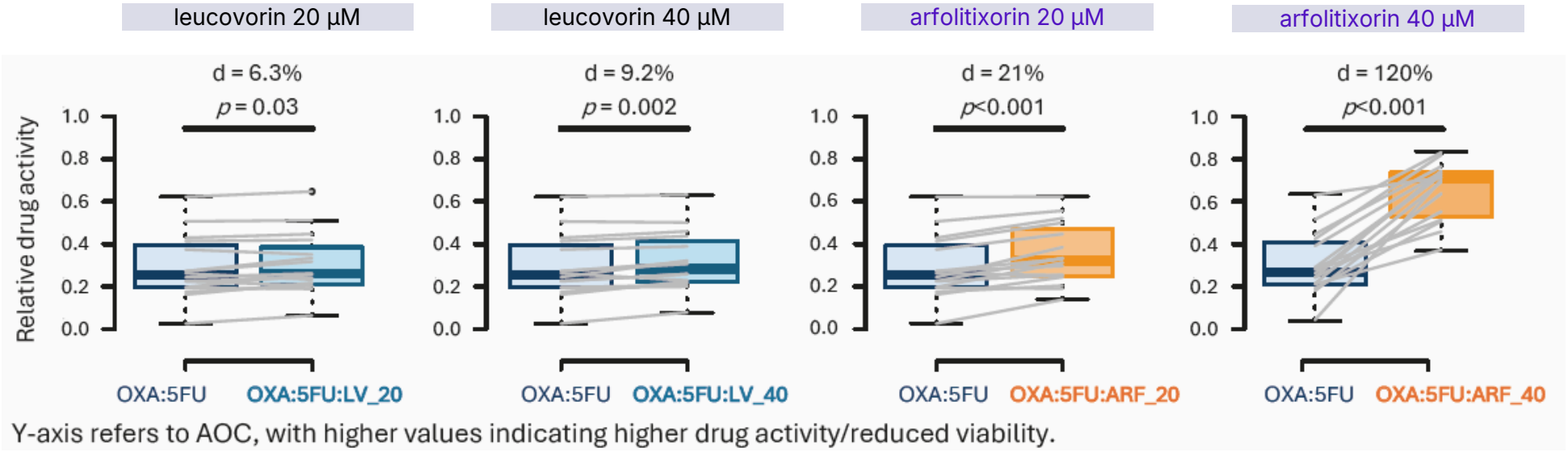
- It is well known that **the clinical efficacy of leucovorin (LV) does not increase with higher doses**. This was reconfirmed in a recent preclinical study where deoxyuridine (dUr) concentrations (a biomarker for TS inhibition as a surrogate for clinical efficacy) were measured in colorectal tumor homogenates.
- For arfolitixorin (Arfo) however, the study showed a strong dose-response relationship, i.e. that **the efficacy of arfolitixorin increased with higher doses**,
- This property is unique to arfolitixorin and distinguishes it markedly from leucovorin, whose efficacy plateaus at higher concentrations

Research by Surgical-Oncology Laboratory, Department of Surgery, Institute of Clinical Sciences, Sahlgrenska University Hospital, Sweden.
E Odin , G Carlsson , B Gustavsson , Y Wettergren , Deoxyuridine as a surrogate marker of thymidylate synthase inhibition contributes to a multivariable model predicting 5-FU/LV response in metastatic colorectal cancer, Cancer Treatment and Research Communications (2025), doi: https://doi.org/10.1016/j.ctarc.2025.101076

iSOFOL

Dose-response: Studies indicate that arfolitixorin's efficacy increases with dose, setting it apart from leucovorin (2/3)

A preclinical study, conducted on Patient-Derived CRC Tumoroids tested arfolitixorin vs. leucovorin in combination with 5-FU and oxaliplatin*, reproduces the dose-response relationship. Arfolitixorin showed potent concentration-dependent cytotoxic effects that enhanced the 5-FU + oxaliplatin activity more effectively than leucovorin. Increased doses potentiated the drug activity even more (+120% with arfolitixorin vs. +9.2% with leucovorin).



Y-axis refers to AOC, with higher values indicating higher drug activity/reduced viability.

d = increase in AOC

Oxaliplatin is a standard component of 5-FU-based chemotherapy regimens

Ref: P. Eide et al. 128P Cytotoxicity of arfolitixorin versus leucovorin (LEU) with 5-fluorouracil (5FU) and oxaliplatin in colorectal cancer (CRC) patient-derived tumoroids (PDTs). Annals of Oncology, Volume 36, Supplement 1, 2025, Pages S56-S57, ISSN 0923-7534, https://doi.org/10.1016/j.annonc.2025.05.140.

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Dose-response: Studies indicate that arfolitixorin's efficacy increases with dose, setting it apart from leucovorin (3/3)

The Modelle-001 trial, conducted on CRC tumor samples from liver metastases from living patients, adds to the body of evidence for the dose-response relationship. Median TS inhibition, a surrogate marker for clinical efficacy, was highest in the group receiving the highest dose of arfolitixorin.

> In conclusion, the Modelle-001 Trial demonstrated significantly higher levels of MeTHF (both mono- and polyglutamates) in metastases following Arfo compared to LV. This resulted in a greater increase TS inhibition in metastases although not statistically significant. All patients in the A30 group showed TS inhibition in metastases, whereas several patients in the LV60 group had no TS inhibition. The median TS inhibition was highest in the A120 group.

A30 = Arfolitixorin 30 mg/m^2. A120 = Arfolitixorin 120 mg/m^2. LV60 = Leucovorin 60 mg/m^2

Ref: Taflin, H. et. al. (2024). Increased potentiation of 5-fluorouracil induced thymidylate synthase inhibition by 5,10-methylenetetrahydrofolate (arfolitixorin) compared to leucovorin in patients with colorectal liver metastases; The Modelle-001 Trial. BJC Reports (2024) Volume 2, Article Number 89. DOI: 10.1038/s44276-024-00111-4

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Arfolitixorin demonstrated comparable efficacy to leucovorin in a previous phase III trial* – despite the use of a suboptimal dosing regimen

Incongruent dosing regimens: 60 mg/m^2 of arfolitixorin given 30 minutes after the 5-FU bolus followed by another 60 mg/m^2 given 30-60 minutes later was compared to 400 mg/m^2 of leucovorin (equimolar to 200 mg arfolitixorin†) given before 5-FU. Two factors seemingly led to that the primary endpoint of superiority was not met:

Wrong timing

Arfolitixorin was given 30 min after the 5-FU bolus, in contrast to leucovorin (control) which was given before as per clinical practice

→ Too late to fully potentiate TS-inhibition as high MTHF levels are required from the start of the formation of the inhibitory complex

Low dose

Arfolitixorin was given in a significantly lower dose than leucovorin

→ Comparing a low dose arfolitixorin with a high dose leucovorin meant
1) an inaccurate comparison between arms, and that
2) arfolitixorin's dose-response relationship was not leveraged

*) The AGENT study was a randomized, controlled, multi-center Phase III study assessing the efficacy and safety of arfolitixorin compared to leucovorin, both used in combination with 5-FU, oxaliplatin, and bevacizumab in first-line metastatic colorectal cancer (mCRC) patients. The endpoint of superiority was not met, and the trial was terminated in 2022.

†) Equimolar to 200 mg arfolitixorin.

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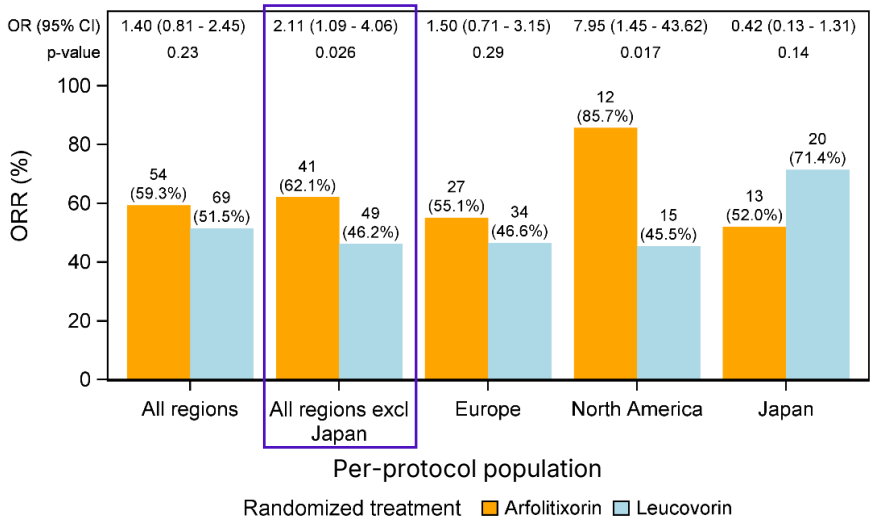
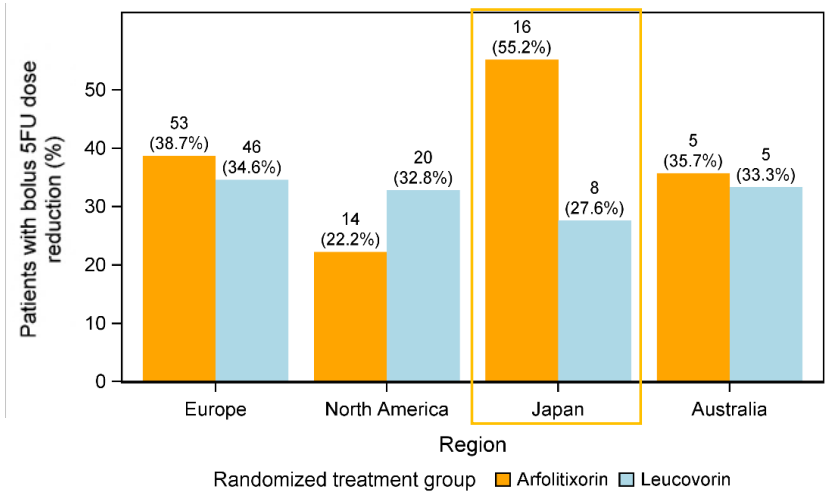
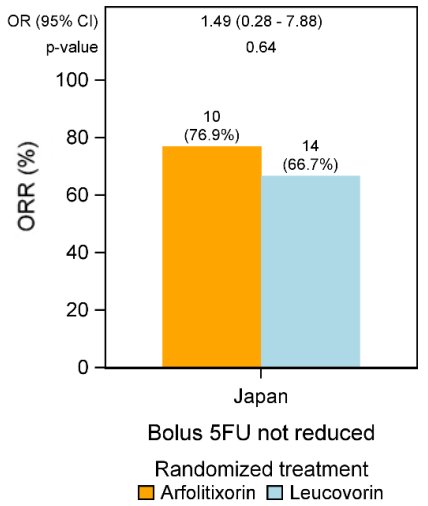
AGENT ph III: Post-hoc, per-protocol analysis indicates possible superior efficacy in all regions excluding Japan even with the suboptimal dosing regimen used







When excluding subjects with study protocol deviations (54% of all patients), arfolitixorin shows **significantly higher efficacy** compared to leucovorin, in all regions excluding Japan.

Significantly higher proportion of reduced 5-FU doses in Japan compared to other geographical regions, which may explain the lower response rates.

Result cleared from subjects with reduced 5-FU doses show numerically higher efficacy also in Japan.

•Aldina Pivodic et al.The importance of treatment handling and compliance on overall response rate in a phase III study of metastatic colorectal cancer: Post-hoc per protocol analyses of the AGENT trial.. *J Clin Oncol* **43**, 205-205(2025).DOI:10.1200/JCO.2025.43.4_suppl.205

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In summary, earlier studies form a comprehensive dataset that de-risks the continued development

CLINICAL SAFETY, EFFICACY

ISO-CC-002 (Phase I/II)

ISO-CC-005 (Phase I/II)

- Phase I/II-studies ISO-CC-002 and ISO-CC-005 indicate that the drug candidate is **safe, well tolerated and show efficacy**.
- It also shows **significantly higher levels of MTHF in tumors** following equimolar doses of arfolitixorin compared to SoC.

CLINICAL RANDOMIZED SAFETY,EFFICACY

AGENT (Global phase III)

AGENT post-hoc analyses

- Global, randomized phase III-study did not meet its endpoint of superiority but **showed that the drug is efficacious** with similar efficacy as leucovorin in the ITT population with the chosen dosing regimen
- Post-hoc analyses show that 1) the **dosing was likely too low** and not comparable to the control arm, and 2) that **the dose was given too late**
- Post-hoc, per-protocol analysis indicates **possible superior efficacy in important regions even with the suboptimal dosing**

PRECLIN/CLIN INDICATING DOSE-RESPONSE

Patient-Derived Tumoroid studies

Tumor homogenate studies

Tissue-level studies

- Recent preclinical studies strengthen the evidence and underpins the restart of the clinical program, e.g. by indicating strong drug activity and pointing at a **strong and unique dose-response relationship** (higher doses of arfolitixorin gives higher efficacy, in contrast to leucovorin).
- This dose-response relationship was not leveraged in AGENT as a low dose was used

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Taken together, the data form a solid evidence platform for continued clinical development

1. **Established efficacy: Arfolitixorin has already shown efficacy comparable to SoC** in a global phase III-study with suboptimal dosing (too low dose given too late).

2. Available data indicate a strong dose-response relationship and that **higher doses given before instead of after 5-FU will lead to better efficacy**.

3. **Safety with higher doses has been established** (up to 500 mg/m^2, which is the highest dose to be tested in the ongoing study) in healthy volunteers.*

*) Studies have been conducted with doses up to 500 mg/m^2 (in healthy volunteers) and 240 mg/m^2 (in patients with metastatic colorectal cancer in combination with 5-FU and other drugs, ARF given in the phase III dosing sequence) with a maintained safety profile.

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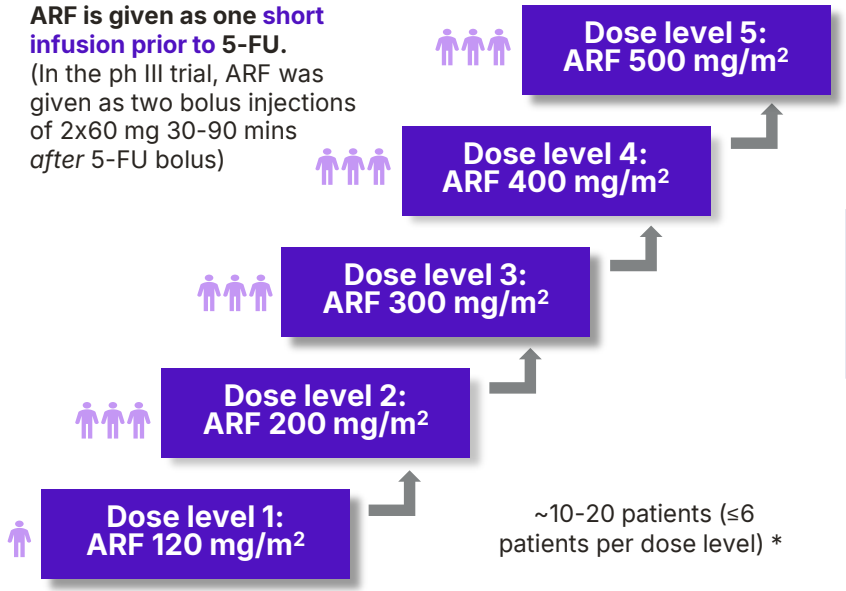
Arfolitixorin phase Ib/II-study

Enrolling 1st line mCRC patients to be treated with standard chemotherapy regimens (ph 1b: FOLFOX+bev, ph 2: FOLFOX/FOLFIRI+bev/cet/pan) where ARF replaces LV in the experimental arms

Ongoing phase 1b 2025-2026:
Dose escalation. RAS-mutant patients

ARF is given as one short infusion prior to 5-FU.
(In the ph III trial, ARF was given as two bolus injections of 2x60 mg 30-90 mins *after* 5-FU bolus)



~10-20 patients (≤6 patients per dose level) *

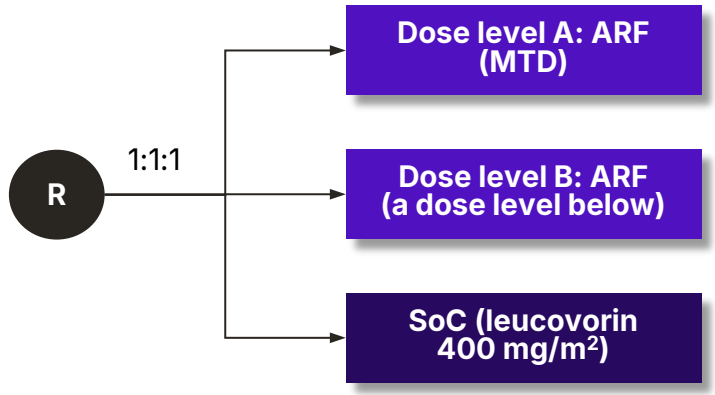
Two doses selected from phase 1b for optimization

Planned phase 2 (randomized) 2026-2027:
Dose optimization. RAS mut and RAS/BRAF WT.

~60 patients in Europe

~20 patients in Japan

Preliminary design, to be confirmed



Key objectives and endpoints	
Primary:	Safety, tolerability
Secondary:	ORR, PFS, DoR, OS

Key objectives and endpoints	
Primary:	Safety, tolerability; ORR, DoR
Secondary:	PFS, TTR, OS, DCR, DpR

*Treatment administered until disease progression, undue toxicity, or any other protocol-defined stopping criterion following a BOIN, Bayesian optimal interval design.
ARF= arfolitixorin; LV= leucovorin; ORR= Objective Response Rate; PFS = Progression Free Survival; DoR= Duration of Response; OS= Overall Survival; TTR= Time to Response; DCR= Disease Control Rate; DpR= Depth of Response

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RECENT PRESS RELEASE

Arfolitixorin phase Ib/II-study – promising early interim readout

- To date, **no dose-limiting side effects** have been observed in the treated patients.
- Preliminary results show that **all patients** included in the study to date **have responded to treatment and exhibited tumor shrinkage**, with total tumor burden reductions of up to approximately 50 percent.
- Half of the six patients evaluated to date have responded so well to the treatment that they were removed from the study for **consideration of tumor surgery** (removal of the tumors), which is unexpected in this RAS-mutated, difficult-to-treat patient population where surgery is not normally considered feasible.

Clinical development in collaboration with Charité – a world leading hospital

CHARITÉ
UNIVERSITÄTSMEDIZIN BERLIN

Prof. Dr. med.
Sebastian Stintzing

Head of the Department of Hematology, Oncology and Cancer Immunology (CCM)

Coordinating Principal investigator

iSOFOL

Isofol's Advisory Board consists of leading global experts representing the US, Europe and Japan

Sebastian Stintzing

Prof. Dr. med. MD Professor
Head of the Clinic for Hematology, Oncology and Cancer Immunology at Charité Universitätsmedizin in Berlin, Germany

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Frits Peters

Professor
Professor emeritus at the Laboratory of Medical Oncology, Amsterdam University Medical Center; Professor at the Medical University of Gdansk, Poland, and honorary Professor of Amity University i Noida, Indien.

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Clinical development and commercialization in Japan in collaboration with partner Solasia

Isofol has licensed the rights to develop and commercialize arfolitixorin in Japan – one of the world's biggest pharmaceutical markets – to Solasia Pharma K.K., a company listed on the Tokyo stock exchange working to bring innovative treatments to Japan and other countries in Asia.

License agreement for development and commercialization

- Under a license agreement, Isofol is entitled to a double-digit royalty compensation based on net sales in Japan, as well as an upfront payment and milestone payments linked to development, regulatory, and sales-based targets.

Investments in clinical development and regulatory activities

- In the spring of 2025, Solasia announced its intention to invest approximately SEK 140 million in the upcoming clinical phase II and III studies of arfolitixorin in Japan, as well as in regulatory approval applications, financing a large part of the development costs in Japan.
- The work is being conducted in close collaboration with Isofol, which supports the Japanese development program and ensures that it aligns with development activities elsewhere and benefits regulatory processes in other geographic regions.

Partnership solidified by shareholding

- In addition to investments in clinical development, Solasia established in 2025 a shareholder position in Isofol, representing an approximate 2% ownership stake as of September 30, 2025.

Isofol has CMC and large scale GMP manufacturing in place – key partnership with Merck KGaA

DRUG SUBSTANCE / API

Merck KGaA Life Science owns and manufactures arfolitixorin – Isofol has global, exclusive rights for development and commercialization in oncology

- Strategic partnership between Isofol and Merck KGaA Life Sience
- Composition of Matter / Drug Substance, production process and Drug Product patented by Merck
- Drug Substance / API production by Merck
- All use patents (clinical use / dosing regimens patented by Isofol

DRUG PRODUCT

Large-scale Drug Product manufacturing secured with Recipharm

- Several large-scale GMP batches completed and released for clinical studies

Isofol's value creation rests on three solid pillars

01

High unmet medical need

02

High-potential drug candidate

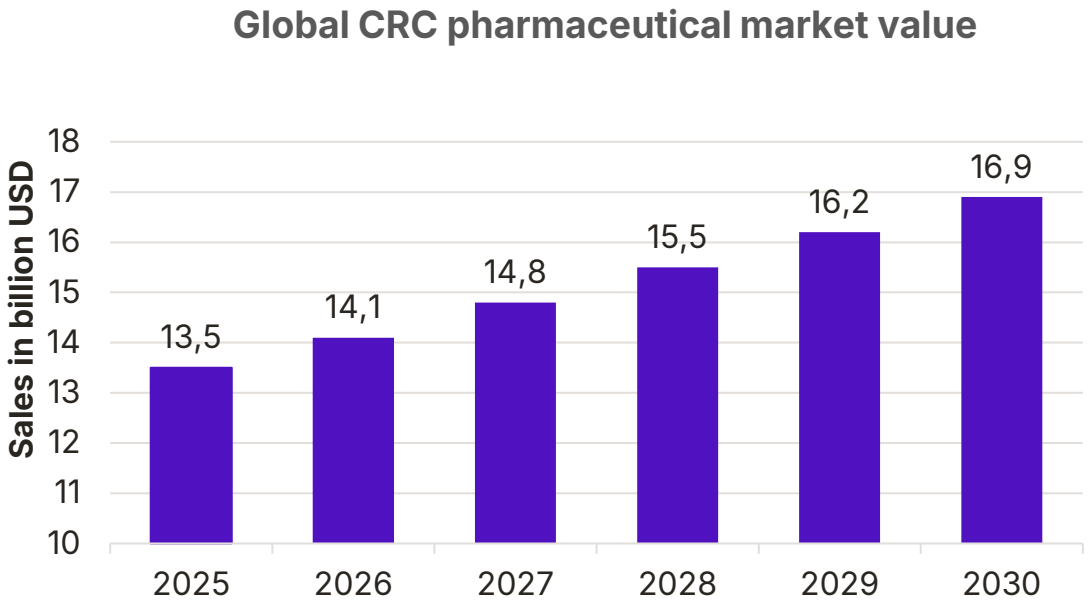
03

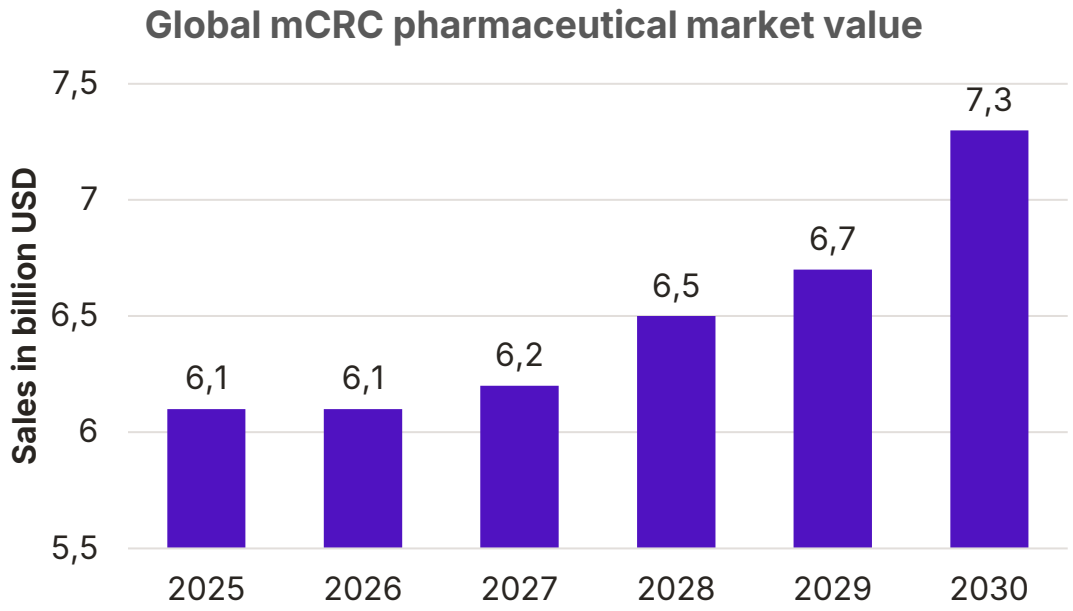
Large market opportunity

Arfolitixorin has blockbuster potential in the US in the lead indication alone – on a global CRC market estimated to be worth more than $17 billion by 2030. Additional indications and markets could add to the opportunity.

Large and growing market for CRC treatments as well as for the lead indication mCRC

The global market for CRC treatments is valued at $13.5 B today and is expected to grow to $16.9 B in 2030; whereas the mCRC market is expected to grow from $6.1 B in 2025 to $7.3 B in 2030.





Sources: mCRC: Back Bay analysis, Evaluate. CRC: Average of various published market analyses.

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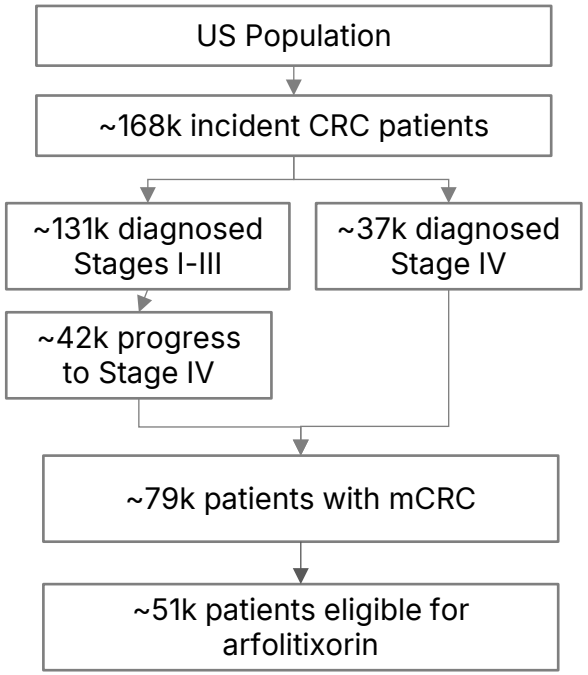
Blockbuster revenue potential in the U.S. in the lead indication mCRC alone

Patient flow (projected 2040):



Key assumptions:

- Comparable safety as SoC
- Efficacy benefit of >15% absolute increase in ORR compared to SoC
- Pricing: Premium to SoC
- Market penetration >50%
- Length of treatment: 8 months
- Treatment compliance: 90%

1

billion USD

Gross yearly sales potential for arfolitixorin in the USA in the mCRC indication alone

- blockbuster potential -

Source: Back Bay analysis, expert interviews; Nov 2024 (updated Dec 2025)

Further opportunities beyond the lead indication in the US add to the potential as additional markets are added and the clinical use expands

1

billion USD

Gross sales potential for arfolitixorin in the USA in the mCRC indication alone

- blockbuster potential -

+

Additional markets ex-U.S.
Incl. e.g. **Japan, Canada,** Europe, Middle East, Asia.

Licensing partnerships already established in Japan (Solasia Pharma K.K) and Canada (Knight Therapeutics)

Solasia

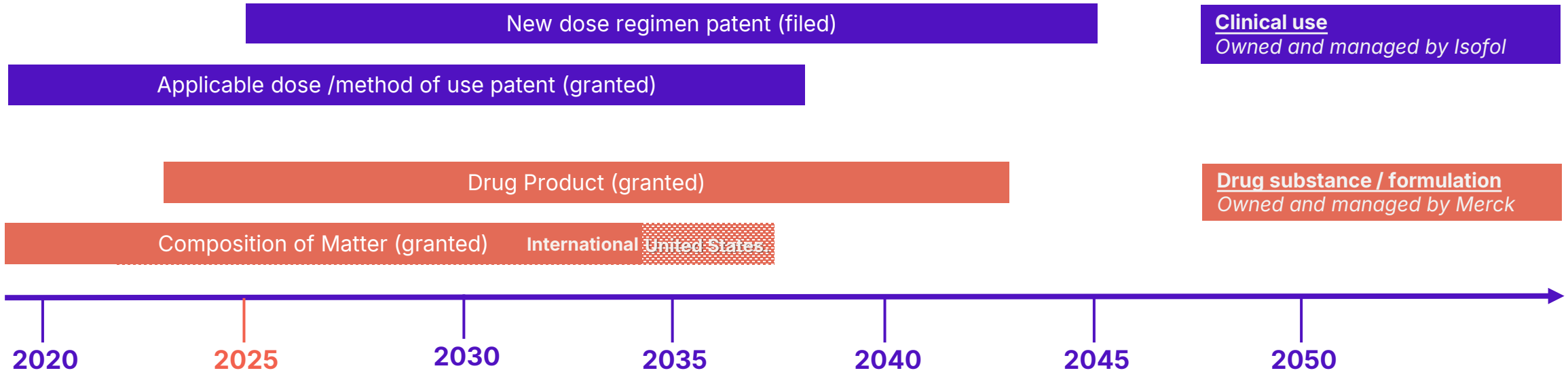
Knight

Additional indications
Adjuvant /neoadjuvant CRC,
other solid tumors where 5-FU + folates are used
(e.g. pancreatic, gastric, breast, head/neck)

Source: Back Bay analysis, expert interviews; Nov 2024

Suite of patents provide for strong intellectual property protection

- Both Isofol and Merck KGaA are actively working to protect and enhance arfolitixorin's suite of patents on the major markets (including but not limited to USA, Europe, Japan)
- **Current patent portfolio (allowed/granted + new filings):**



Isofol's value creation rests on three solid pillars

01

High unmet medical need

Arfolitixorin aims to potentiate 5-FU-based chemotherapy – todays and tomorrow's standard treatment for several types of cancer where better treatments are urgently needed

02

High-potential drug candidate

Arfolitixorin is the first and only direct-acting folate, designed to enhance 5-FU efficacy and improve outcomes of standard treatments across multiple cancer types. It has shown promising results in earlier studies.

03

Large market opportunity

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Management Team

Petter Segelman Lindqvist
Chief Executive Officer

Petter Lindqvist holds an MSc in Business Administration from the Stockholm School of Economics and EM Lyon. He has held leadership roles at GSK, Abbott, AbbVie and Sobi, and has experience from biotech board positions. His background spans strategic business development, global commercialization, and guiding drug candidates through clinical development and regulatory approval. He joined Isofol in 2024.

Dr Roger Tell, MD, PhD
Chief Medical Officer

Dr Roger Tell, MD, PhD is a board-certified oncologist affiliated with Karolinska University Hospital and Karolinska Institutet. He joined Isofol in 2019 and currently leads the company's medical and scientific strategy. Dr. Tell previously held senior leadership roles at Aprea Therapeutics and Servier, and he has extensive experience as a practicing oncologist and strategic advisor to several leading global biopharmaceutical companies, including Eli Lilly, AstraZeneca, and Merck Serono. He also serves as a member of the Board of Directors of Vivesto AB, a company listed on Nasdaq Stockholm.

Margareta Hagman
Chief Financial Officer

Margareta Hagman is an experienced CFO with senior financial leadership roles, mainly at BioGaia AB, but also at Xbrane Biopharma AB and Ortivus AB and is member of the Board of Directors of Infant Bacterial Therapeutics AB – all companies listed on Nasdaq Stockholm. Margareta joined Isofol in 2024, bringing extensive expertise from listed life science companies.

abbvie

AstraZeneca

sobi

Lilly

SERVIER
moved by you

BioGaia

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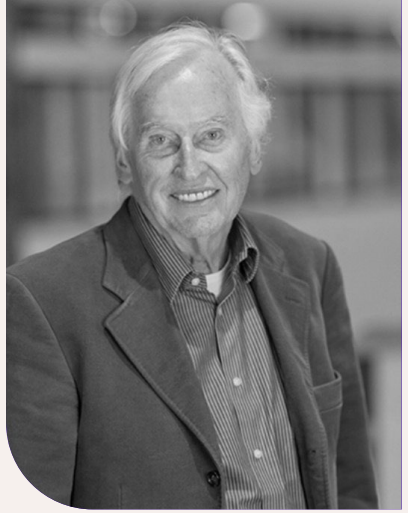
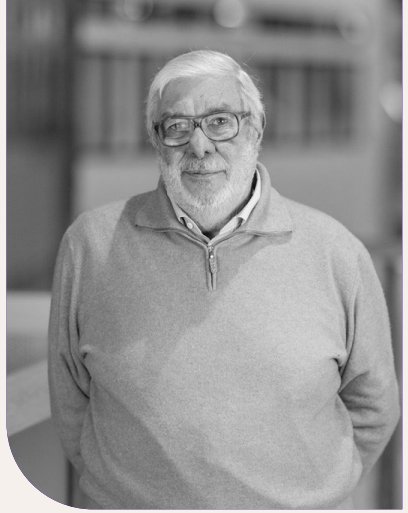


Jan-Eric Österlund
Chairman of the Board

Jan-Eric Österlund has decades of experience in private equity and management buy-outs with a strong focus on life sciences. He has served as CEO, director or chairman in public companies across the US, Canada, Switzerland and Sweden. He is based in England and brings deep strategic and international board expertise to Isofol.



Dr Alain Herrera
Board Member

Dr Alain Herrera is an oncologist/hematologist who played a key role in the global registration of oxaliplatin, a cornerstone of modern colorectal cancer therapy (FOLFOX). He has held senior global oncology leadership roles at Sanofi and other pharmaceutical companies and currently serves as a senior consultant and member of several supervisory boards in oncology.

Dr Helena Taflin
Board Member

Dr Helena Taflin is an Associate Professor of Surgery specializing in liver surgery and transplant at Sahlgrenska University Hospital, where she also heads the Clinical Trial Unit. Her research focuses on folate metabolism in colorectal cancer, and she maintains an active role in clinical studies and national medical associations.

Lars Lind
Board Member

Lars Lind serves on Isofol's Compensation, Audit, and Nominating Committees. His background includes longstanding experience from executive positions in large, international public companies as well as senior board experience, contributing with business strategy, governance and oversight capability to the company. Mr Lind was instrumental in the founding of Isofol and has served on the board for several years.

Prof Sten Nilsson, MD
Board Member

Prof Sten Nilsson is an experienced oncologist and professor at the Karolinska Institute and independent board member bringing deep clinical and academic expertise to Isofol. With a distinguished career spanning clinical oncology and cancer drug development, he has contributed to the development of major therapies.

Board of Directors

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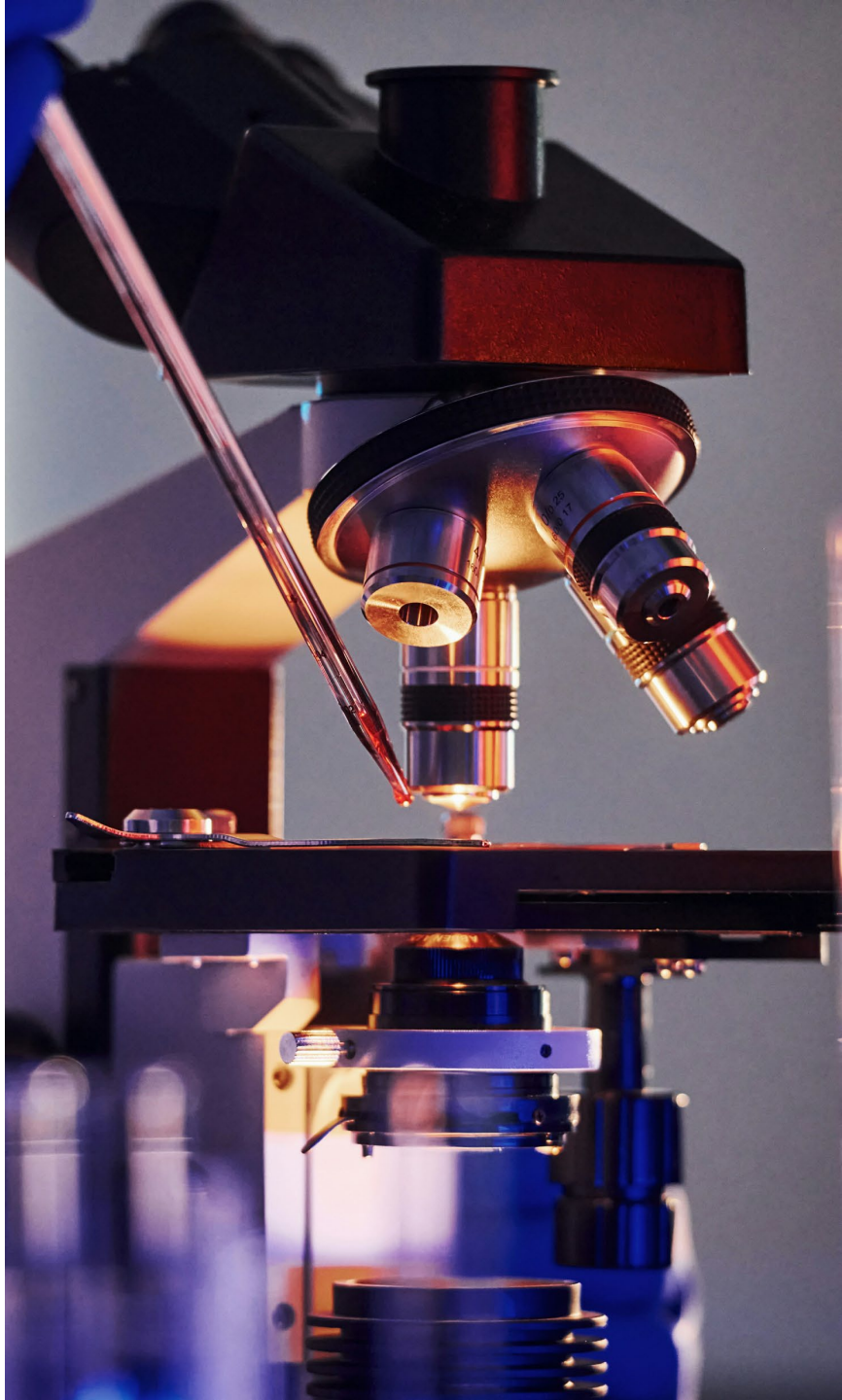
The Isofol opportunity in short

- Focusing on areas in oncology of high unmet medical need
- Large amount of data available: de-risking development. CMC and large-scale manufacturing established.
- Next-gen version of an established mechanism of action with widespread clinical use: facilitates market adoption
- Blockbuster potential, favorable competitive landscape and strong IP protection
- Solid partner network and strong team in place to drive flawless and swift execution

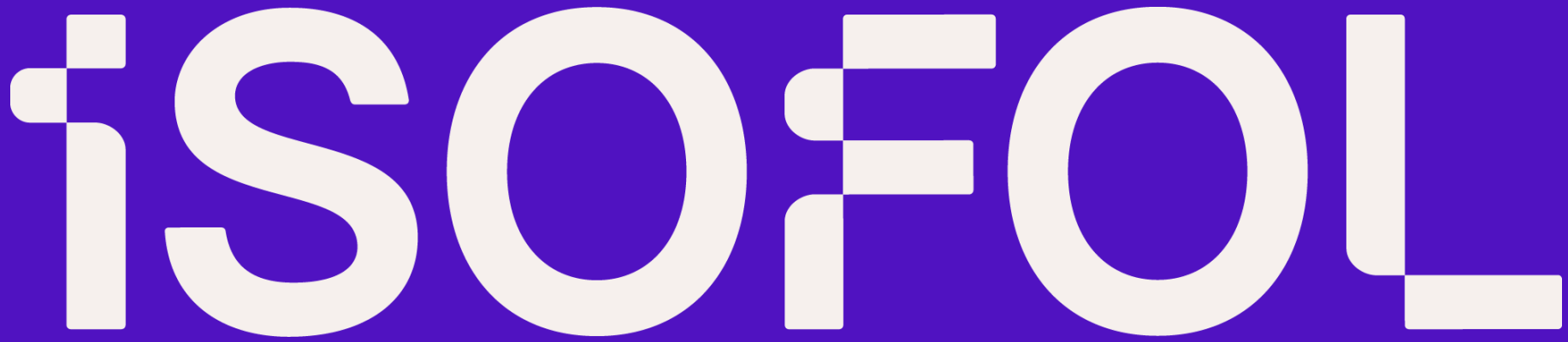


Our goal

We aim to have a central impact on tomorrow's cancer treatment by giving millions of patients the opportunity to respond better to their treatment, improve their prognosis, and gain more time with life.

By this, we strive to create significant value for patients and their families, healthcare providers, shareholders and partners – and ultimately for society at large.

iSOFOL



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